Relationship Between Postoperative Lordosis Distribution Index And Adjacent Segment Disease Following L4-S1 Posterior Lumbar Interbody Fusion

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Abstract

Background. ASD is an acknowledged problem of PLIF. Many studies have been reported concerning the role of LDI in spinal biomechanics. However, few reports have been published about the impact of LDI on ASD following L4-S1 PLIF.

Methods. The study enrolled 200 subjects who underwent L4-S1 PLIF for degenerative spine disease from 2009 to 2014. The average follow-up term was 84 months. Several lower lumbar parameters were measured, including lower lumbar lordosis (LLL), lumbar lordosis (LL) and LDI on the pre- and postoperative radiograph. Perioperative information, comorbidities and operative data were documented. Kaplan-Meier curves were plotted for the comparisons of ASD-free survival of 3 different kinds of postoperative LDI subgroups.

Results. The incidence of ASD was found to be 8.5%. LL and LLL increased by 3.96 ° (38.71 ° vs 42.67 ° , P ⏃ 0.001) and 3.60 ° (26.22 ° vs 28.82 ° , P ⏃ 0.001) after lower lumbar fusion surgery, respectively. Lordosis distribution index (LDI) increased by 0.03 (0.66 vs 0.69, P=0.004) postoperatively. A significant difference(P=0.001) was observed when comparing the incidence of ASD among postoperative LDI subgroups. The Kaplan-Meier curves showed a marked difference in ASD-free survival between low and moderate LDI subgroup (Log Rank test, P=0.0012) , high and moderate LDI subgroup (Log Rank test, P=0.0005)

Conclusion. Patients with abnormal postoperative LDI were statistically more likely to develop ASD than those who had normal postoperative LDI. Moreover, patients with low postoperative LDI were at greater risk for developing ASD than those with high postoperative LDI over time.

Introduction

Posterior lumbar interbody fusion (PLIF) with pedicle screw fixation has been widely accepted to treat various lumbar spinal disorders involving lumbar spinal stenosis, lumbar disc herniation, degenerative spinal deformity, instability, spondylolisthesis and vertebral compression fracture. Although PLIF with pedicle screw fixation could provide solid instrumentation and achieve satisfactory clinical effects, it may alter the normal biomechanics of the spine and accelerate the degenerative process of adjacent unfused segments, resulting in adjacent segment disease.[1-6] Adjacent segment disease (ASD) is one of the widely acknowledged problems of posterior lumbar interbody fusion which encompasses many complications, including herniated nucleus pulposus, spondylolisthesis, stenosis, hypertrophic facet arthritis and scoliosis.[5,7,8]

Various studies have proposed the risk factors for the development of ASD after lumbar spinal fusion, including age, sex ,obesity, body mass index(BMI),fusion length, osteoporosis, laminectomy performed adjacent to a segment, preoperative segmental instability at the adjacent level, preexisting degenerated disc prior to fusion and excessive disk height distraction.[9-12] In addition, lower lumbar interbody fusion

surgeries constitute the main part of the spinal surgeries in a spine surgeon's clinical work.[13] The total lumbar lordosis mainly consists of the upper-arc lordosis of L1-L3 and lower-arc lordosis of L4-S1. Lower lumbar lordosis(LLL), defined as the angle between the superior endplate of L4 and S1, accounts for two thirds of the total lumbar lordosis.[14,15] Lordosis distribution index (LDI), described as L4−S1 lordosis/L1−S1 lordosis × 100%, defines the magnitude of lower-arc lordosis relative to the total lordosis.[16] Few studies have shown the impact of LDI on ASD after lower lumbar spine surgery. Therefore, it is critical to explore the factors to reduce the incidence of ASD in patients performed lower lumbar spine.

The aim of this study is to examine the association of postoperative LDI with ASD following L4-S1 posterior lumbar interbody fusion with pedicle screw fixation for spinal degenerative diseases, and provide guidance in surgical planning for a spine surgeon to improve patients' outcome and further alleviate medical costs.

Materials And Methods

Subjects and Surgical Procedure

This is a retrospective study, including a total of 215 consecutive patients who were treated for spinal degenerative pathologies at the Chinese PLA General Hospital over a 5-year period from 2009 to 2014. The protocol was approved by the research ethics committee of the hospital. The patients were enrolled to meet the following inclusion criteria: degenerative disorders of lower lumbar spine such as lumbar disc herniation and symptomatic lumbar spinal stenosis with severe lower back pain and radiculopathy. All the subjects were performed with L4-S1 PLIF and bilateral lumbar laminectomy of vertebrae L4-5 using an PEEK cage inserted to intervertebral space. The cage was placed after the disk space was cleaned and the autograft bones crumbled from resected lamina were put into the space. The minimum follow-up period was 19 months with imaging collection including lumbar plain anteroposterior and lateral X-ray obtained before surgery, immediately following surgery. CT and MRI scans were obtained before surgery, the instant a new onset of symptoms appeared and at the final follow-up visit. We excluded patients who were diagnosed as spinal neoplasm, trauma, hip joint disease, infection, compression fracture of vertebra, inflammation, rheumatoid arthritis and isthmic spondylolisthesis. Subjects who had scoliosis with a Cobb angle≥10°, previous lumbar fusion or laminectomy surgeries, preoperative spondylolisthesis≥3 mm at the adjacent level and severe adjacent intervertebral disc degeneration of Pfirrmann Classification V were also excluded from this study.[17] Therefore, 200 patients (89 men and 111 women) were enrolled in this study with a mean age of 64.8±8.5 years (range, 36 years to 85 years). The average follow-up term was 84 months (P$_{25}$–P$_{75}$, 70–98 months).

Adjacent segment disease (ASD) is defined as a pathologic condition seen on radiographs with clinical symptoms in which an additional surgical intervention may be required to treat neurological symptoms at the level adjacent to previous fusion.[18] The radiographic ASD was defined by segmental kyphosis more than 10°, the development of anterolisthesis or retrolisthesis of more than 3mm and a deterioration in the Pfirrmann classification(grades I−V) or Imagama's Classification (grades I−IV) of one-grade or greater

progression at the level adjacent to a previous spinal fusion.[19-21] The clinical ASD is considered to be adjacent disc degeneration leading to newly developed symptoms, such as stenosis, instability and neurological abnormality.[22]

Radiographic Evaluation and Clinical Data Collection

Lumbar lordosis(LL) is defined by the angle between the superior endplate of L-1 and S-1 on the neutral lateral X-ray image. Lower lumbar lordosis(LLL) is measured from the superior endplate of L4 to the superior endplate of S1 by the Cobb method on lumbar plain lateral X-ray. Lordosis distribution index (LDI) is represented by the following formula: lower lumbar lordosis/lumbar lordosis × 100% (Figure 1). All measurements were performed 2 times by a trained spine surgeon blinded to clinical outcomes with the aid of Surgimap (version 2.3),and the mean values were recorded for the analysis. LDI is divided into 3 subgroups. An LDI less than 50% is considered hypolordotic maldistribution (low LDI); 50%–80%, aligned (moderate LDI); and more than 80%, hyperlordotic maldistribution (high LDI).[16] Perioperative information including demographic variables, comorbidities and operative data.

Statistical Analysis

Continuous variables are presented as meanstandard deviation or P_{50} (P_{25}⊠P_{75}) according to Shapiro-Wilk normality tests. SPSS (version 23) for windows was performed for statistical analysis. The Pearson $\chi 2$ test or Fisher's exact test was used for the comparison of sex, BMI intergroup, comorbidities, pre and postoperative LDI subgroup between ASD and Non-ASD group. The nonparametric test of Mann-Whitney U test was applied for follow-up period, intraoperative blood loss, operation time and hospital stay. The independent-samples t test was used for radiographic parameters of the lumbar spine. The paired-samples t test was used to compare pre and postoperative changes in the lower lumbar parameters. Kaplan-Meier curves were plotted and compared by the use of log-rank tests. An α level of 0.05 was considered to be statistically significant.

Results

Subjects and Surgical Procedure

Adjacent segment disease (ASD) after L4-S1 PLIF for degenerative disease of lower lumbar spine was developed in 17 of 200 patients (8.5%) at the final follow-up. Segment lesions of them were identified at the level above the fusion and all of them were enrolled in the ASD group. In the ASD group, 6 male and 10 female subjects were included, with an average age of 66.1±5.1.The mean follow-up period was 62.8±24.6 months. BMI at admission was 27.04±3.00 kg/m^2 at average. In the Non-ASD group, 82 male and 101 female subjects were included, with an average age of 64.7±8.7.The average follow-up duration

was 85 months (72 months～99 months). BMI at admission was 23.87±3.33 kg/m^2 at average. 101 patients had BMI≥25 kg/m^2, 13 patients (12.87%) of which finally developed ASD. 121 patients had BMI＜ 25 kg/m^2, 4 patients (3.31%) of which were diagnosed as having ASD in the end. Table 1 show the comparisons of basic characteristics of patients between ASD and Non-ASD group. Statistical analysis identified the significant difference in BMI (23.87 vs 27.04, p＜0.001) , BMI intergroup (23.5% vs 76.5%, P=0.001) and the average follow-up period (85.0 vs 62.8, P＜0.001). But there was no statistically significant difference between the two groups in such basic variables as age, sex , comorbidities, hospital stay, intraoperative blood loss and operation time. Figure 2 illustrates the frequency distribution of follow-up time of ASD patients after L4-S1 PLIF.

Radiographic Evaluation and Clinical Data Collection

In the radiographic evaluation, measured lower lumbar spinal parameters using pre and postoperative radiographs are shown in table 2.LL and LLL increased by 3.96°(38.71° vs 42.67°, P＜0.001) and 3.60°(26.22° vs 28.82°, P＜0.001) after lower lumbar fusion surgery, respectively. Moreover, LDI increased by 0.03 (0.66 vs 0.69, P=0.004) postoperatively. Table 3 demonstrates the comparisons of lower lumbar spinal parameters and LDI with and without ASD groups. There was no statistical significance in radiographic parameters of pre LL(37.17° vs 38.85°,P=0.341), pre LLL(23.71° vs 26.36°,P=0.177), the value of pre LDI(0.65 vs 0.66,P=0.682), post LL(40.35° vs 42.88°,P=0.241), post LLL(27.69° vs 28.93°,P=0.410), and the value of post LDI (0.71 vs 0.68, P=0.578). When comparing pre and postoperative LDI subgroups between ASD and Non-ASD groups, the patients with low LDI pre and postoperatively in the ASD group was 2 (11.8%) and 4 (23.5%), respectively. The patients with moderate LDI pre and postoperatively in the ASD group was 13 (76.4%) and 6 (35.3%), respectively. The patients with high LDI pre and postoperatively in the ASD group was 2 (11.8%) and 7 (41.2%), respectively. Consequently, significant difference was found in the postoperative LDI subgroups (P=0.001) but none in the preoperative LDI subgroups (P=0.252).

Relationship Between Postoperative LDI and ASD

   With respect to the abnormal range of LDI that could be associated with the development of ASD, we attempted to integrate the low LDI subgroup and the high LDI subgroup into a single group named abnormal LDI intergroup. The patients with normal LDI pre and postoperatively in the ASD group was 13 (76.5%) and 6 (35.3%), respectively. The patients with abnormal LDI pre and postoperatively in the ASD group was 4 (23.5%) and 11 (64.7%), respectively. Statistical analysis revealed a significant difference between normal and abnormal LDI intergroup postoperatively (P=0.001), but none was found in the preoperative status (P=0.267). A typical case is presented in figure 3. When comparing the incidence of ASD among different postoperative LDI subgroups, we find statistical significance between low and

moderate LDI subgroup (25% vs 4.1%, P=0.006), high and moderate LDI subgroup (18.4% vs 4.1%, P=0.007) but none was observed between low and high LDI subgroup (25% vs 18.4%, P=0.859)(table 4).

Kaplan-Meier analysis survivorship by LDI subgroups was performed to assess the rate of Non-ASD survival for ASD in patients undergoing L4-S1 PLIF (Figure 4). In low LDI subgroup, ASD-free survival was estimated to be 87.5% at 3 years and 75.0% at 6 years. In moderate LDI subgroup, ASD-free survival was estimated to be 99.3% at 3 years and 97.6% at 6 years. In high LDI subgroup, ASD-free survival was estimated to be 97.4% at 3 years and 84.5% at 6 years. The Kaplan-Meier curves showed a marked difference in ASD-free survival among the three kinds of LDI subgroups (Log Rank test, P=0.001). When comparing survival proportions of every two kinds of LDI groups, we found that the survival rate differ significantly between low and moderate LDI subgroup (Log Rank test, P=0.0012) , high and moderate LDI subgroup (Log Rank test, P=0.0005).However, no statistical significance was observed between low and high LDI subgroup(Log Rank test, P=0.7223).

Discussion

Adjacent segment disease (ASD) is a thorny sequelae after posterior lumbar interbody fusion for spinal degenerative diseases.[1-8] Many studies have reported mechanisms concerning the cause of ASD. However, the specific pathogenesis of ASD is still unknown. Liuke et al[23] revealed the fact that BMI ≥ 25 kg/m^2 increases the risk of lumbar disc degeneration. Bagheri et al [24] demonstrated that patients with higher preoperative BMI have a statistically significant increased risk of developing ASD. Our results were consistent with the previous studies. Normal aging and degenerative process could partially accelerate the progression of ASD. Patient age at the time of the index surgery has been identified as one of the most important risk factors for ASD. Several studies have shown that the incidence of ASD tends to be higher according to advancing age.[21,25] But no statistical relationship was observed between advancing age and ASD in our study. The reported incidence of ASD varies widely, ranging from 5.2–18.5%.[4] We reported a cumulative ASD incidence of 8.5%. In this series, we defined ASD as radiographic abnormalities with new clinical symptoms requiring reoperation. The incidence of ASD would be underestimated as some patients may be not elected in the ASD group.

The most important finding of this research was that the lower lumbar spinal parameter (postoperative LDI) would be a significant risk factor of ASD after L4-S1 PLIF. In most studies, LL has been deeply explored in patients with ASD. Djurasovic et al [26] reported that patients who developed ASD have significantly less LL. Nakashima et al [25] concluded that obtaining appropriate LL in PLIF is important for prevention of ASD. Wu [27] reported that the postoperative angle of LL was 7.9° higher than the preoperative angle in patients with degenerative lumbar scoliosis after PLIF. However, few studies focused on construction of LDI influenced by the ratio of LLL and LL. In our study, the angle of LL and LLL increased by 3.96°and 3.60° after surgery, respectively. LDI increased by 0.03 postoperatively. Except for postoperative LDI subgroups, we found no significant association between ASD and preLL, preLLL, the value of preLDI, preoperative LDI subgroups, postLL, postLLL and the value of postLDI.

To the best of our knowledge, this is the first retrospective study to investigate the association between postoperative LDI and ASD after L4-S1 PLIF in which LLL was constant for instrumentation of L4-S1. In a study of 222 patients with degenerative spinal scoliosis, Yilgor et al[16] developed a new method of GAP score, in which LDI is a key component, to analyze the spinopelvic alignment and predict the mechanical complications after adult spinal deformity surgery. It has also reported that the integration of relative lumbar lordosis(RLL) and lordosis distribution index (LDI), compared with PI-LL, may contribute to lower rates of mechanical complications and better long-term HRQOL.[28] Ohba et al [29] concluded that postoperative LDI is critical to the prevention of excess reciprocal progression of TK resulting in proximal junctional kyphosis. These studies serve as a reminder for us spine surgeons of the role of LDI in spinal biomechanics. In this paper, we clarified the relationship between postoperative LDI and ASD after L4-S1 PLIF. The patients with abnormal postoperative LDI (LDI◻0.5, LDI◻0.8) were more likely to develop ASD than those with normal postoperative LDI ($0.5 \leq LDI \leq 0.8$). The incidence of ASD in low LDI group and high LDI group was 25% and 18.4% respectively, significantly higher than that (4.1%) in moderate LDI group. Abnormal LDI may alter the physiological distribution of loads and increase biomechanical stress at the level adjacent to the fused segment, leading to accelerated disc degeneration and intervertebral instability.[30]

Although patients with abnormal LDI had lower ASD-free survival than that in patients with normal LDI, low LDI seems to raise more concerns than high LDI according to Kaplan-Meier survivorship analysis. Menezes-Reis et al[31] reported that hypolordosis of the lumbar spine was associated with a higher frequency of ASD. Postoperative loss of lumbar lordosis has been found to cause excessive mobility and increased biomechanical stress[32]. This in turn causes premature degeneration of the facet joints. As the facet degenerate, translation of the adjacent segment may occur and produce listhesis. This is in accordance with our findings that patients with low LDI were more vulnerable to the disease of ASD.

Our study design had several potential limitations. First, it was a retrospective study which had inherent difficulties in studying ASD. Further prospective studies are needed to validate the relationship between ASD and postoperative LDI. Second, this is a single-center study; hence, the results may not be representative of all patients undergoing L4-S1 PLIF.

Conclusions

This study investigated the relationship between postoperative LDI and ASD in 200 patients treated with L4-S1 PLIF at an average follow-up of 84 months. BMI is a risk factor for ASD in patients undergoing L4-S1 PLIF for degenerative spine diseases. Patients who had abnormal postoperative LDI were statistically more likely to develop ASD than those with normal postoperative LDI. Moreover, patients with low postoperative LDI were at greater risk for developing ASD than those with high postoperative LDI over time, and obtaining appropriate postoperative LDI in L4-S1 PLIF was confirmed to be important for the prevention of ASD.

List Of Abbreviations

LDI: lordosis distribution index; PLIF: posterior lumbar interbody fusion; ASD: adjacent segment disease; LL: lumbar lordosis; LLL: lower lumbar lordosis;BMI: body mass index

Declarations

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Tables

Table 1. Comparisons of basic characteristics of patients between ASD and Non-ASD group

	Total(n=200)	Non-ASD	ASD	P
Age, years	64.8±8.5	64.7±8.7	66.1±5.1	0.315
Sex, n (%)				0.773
Male	89 (44.5)	82 (44.8)	7 (41.2)	
Female	111 (55.5)	101 (55.2)	10 (58.8)	
BMI	24.14±3.42	23.87±3.33	27.04±3.00	**<0.001**
BMI group, n(%)				**0.001**
BMI<25 kg/m^2	121 (60.5)	117 (63.9)	4 (23.5)	
BMI≥25 kg/m^2	79 (39.5)	66 (36.1)	13 (76.5)	
Comorbidities, n (%)	131 (65.5)	119 (65.0)	12 (70.6)	0.645
Hypertension				
Diabetes Mellitus	57 (28.5)	51 (27.9)	6 (35.3)	0.713
Coronary artery disease	97 (48.5)	90 (49.2)	7 (41.2)	0.528
COPD	39 (19.5)	34 (18.6)	5 (29.4)	0.448
Hospital Stay (days)	9.0 (7.0-11.0)	9.0 (7.0-11.0)	8.0 (6.0-11.5)	0.757
Follow-up period (months)	84.0 (70.0-98.0)	85.0 (72.0-99.0)	62.8±24.6	**<0.001**
Intraoperative blood loss(ml)	290(220-380)	290(220-380)	294.7±111.0	0.920
Operation time (hours)	4.45 (3.70-5.30)	4.50 (3.70-5.30)	4.35±0.87	0.525

Mann-Whitney U test, Pearson χ2 test, independent-samples t test
Abbreviation: ASD, adjacent segment disease; BMI, body mass index; COPD, chronic obstructive pulmonary disease

Table 2.Changes in pre and postoperative radiographic parameters

Radiographic Parameters	Pre Op	Post Op	P
Lumbar Lordosis (°)	38.71±6.96	42.67±8.51	**<0.001**
Lower Lumbar Lordosis(°)	25.22±4.82	28.82±5.90	**<0.001**
Lordosis Distribution Index	0.66±0.10	0.69±0.13	**.004**

Paired-samples t test

Table 3. Comparisons of lower lumbar spinal parameters and LDI in the different groups

	Total	ASD	Non-ASD	P
Pre LL (°)	38.71±6.96	37.17±7.31	38.85±6.93	0.341
Pre LLL (°)	25.22±4.82	23.71±4.55	25.36±4.83	0.177
Pre LDI	0.66 (0.60-0.71)	0.65±0.11	0.66 (0.60-0.71)	0.682
Pre LDI Group (Low LDI, Moderate LDI, High LDI),n(%)				0.252
Low LDI⁬0.5	13 (6.5)	2 (11.8)	11 (6.0)	
0.5≤Moderate LDI≤0.8	172 (86.0)	13 (76.4)	159 (86.9)	
High LDI⁬0.8	15 (7.5)	2 (11.8)	13 (7.1)	
Pre LDI Group (Normal LDI, Abnormal LDI) n(%)				0.267
0.5≤Normal LDI≤0.8	172 (86.0)	13 (76.5)	159 (86.9)	
Abnormal LDI(⁬0.5,⁬0.8)	36 (14.0)	4 (23.5)	24 (13.1)	
Post LL (°)	42.67±8.51	40.35±7.96	42.88±8.55	0.241
Post LLL (°)	28.82±5.90	27.69±5.68	28.93±5.93	0.410
Post LDI	0.69±0.13	0.71±0.19	0.68±0.12	0.578
Post LDI Group (Low LDI, Moderate LDI, High LDI),n(%)				0.001
Low LDI⁬0.5	16 (8.0)	4 (23.5)	12 (6.6)	
0.5≤Moderate LDI≤0.8	146 (73.0)	6 (35.3)	140 (76.5)	
High LDI⁬0.8	38 (19.0)	7 (41.2)	31 (16.9)	
Post LDI Group (Normal LDI, Abnormal LDI) n(%)				0.001
0.5≤Normal LDI≤0.8	146 (73.0)	6 (35.3)	140 (76.5)	
Abnormal LDI(⁬0.5,⁬0.8)	54 (27.0)	11 (64.7)	43 (23.5)	

Independent-samples t tests, Mann-Whitney U test, Pearson χ2 test, Fisher's exact test

Abbreviation: ASD, Adjacent Segment Disease; LL, Lumbar Lordosis; LLL, Lower Lumbar Lordosis; LDI, Lordosis Distribution Index

Table 4. Comparisons of incidence of ASD among different postoperative LDI groups.

	Total (n)	Non-ASD	ASD	P
Low LDI, n(%)	16	12 (75)	4 (25)	.006
Moderate LDI, n(%)	146	140(95.9)	6 (4.1)	
Low LDI, n(%)	16	12 (75)	4 (25)	.859
High LDI, n(%)	38	31(81.6)	7(18.4)	
Moderate LDI, n(%)	146	140(95.9)	6 (4.1)	.007
High LDI, n(%)	38	31(81.6)	7(18.4)	

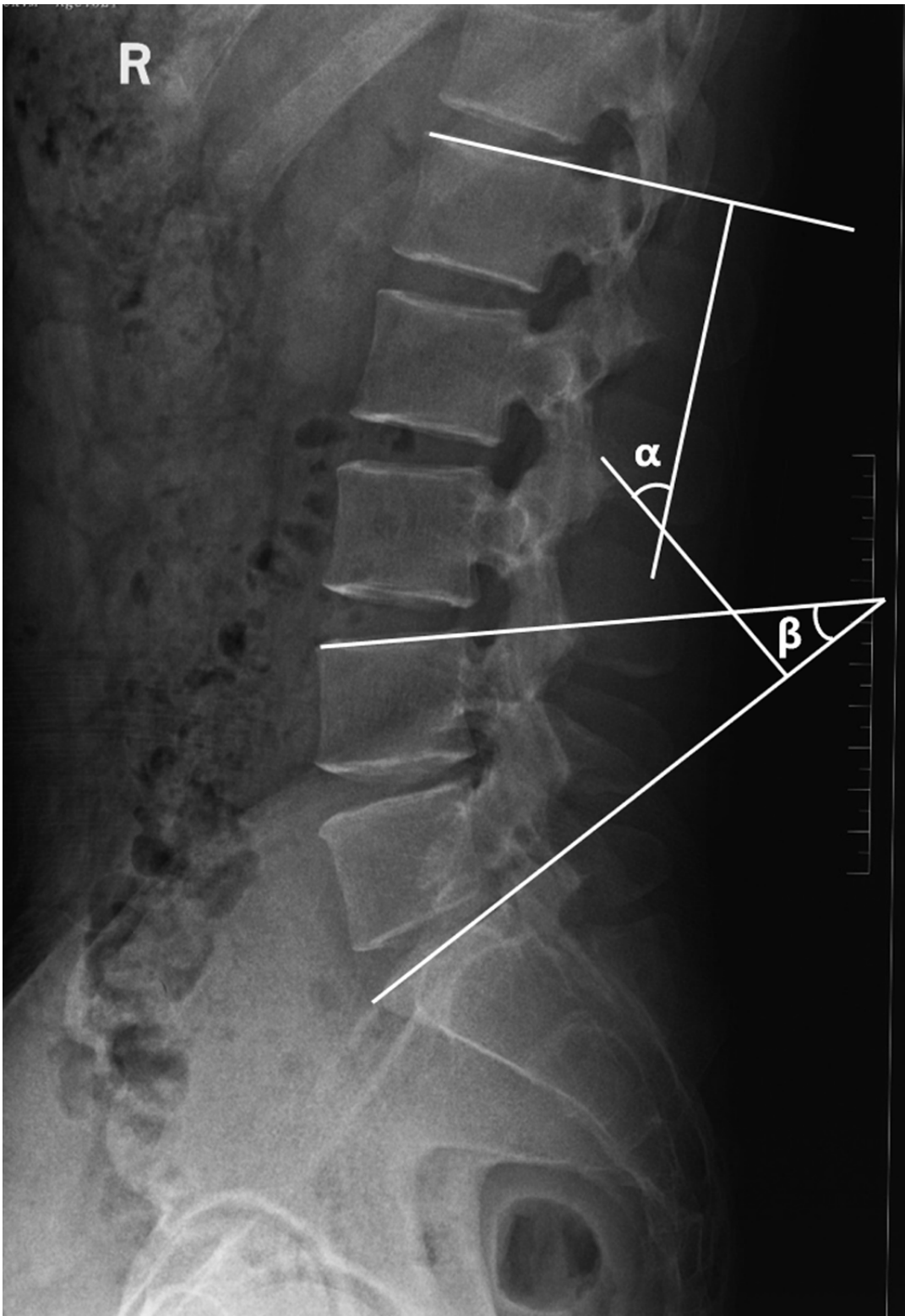Abbreviation: ASD, Adjacent Segment Disease; LDI, Lordosis Distribution Index

Figures

Figure 1

Measurement of lower lumbar spinal parameters. LL(α) is the angle between the superior endplate of L-1 and S-1. LLL(β) is the angle between the superior endplate of L4 and S1. LDI equals to β/α× 100%.
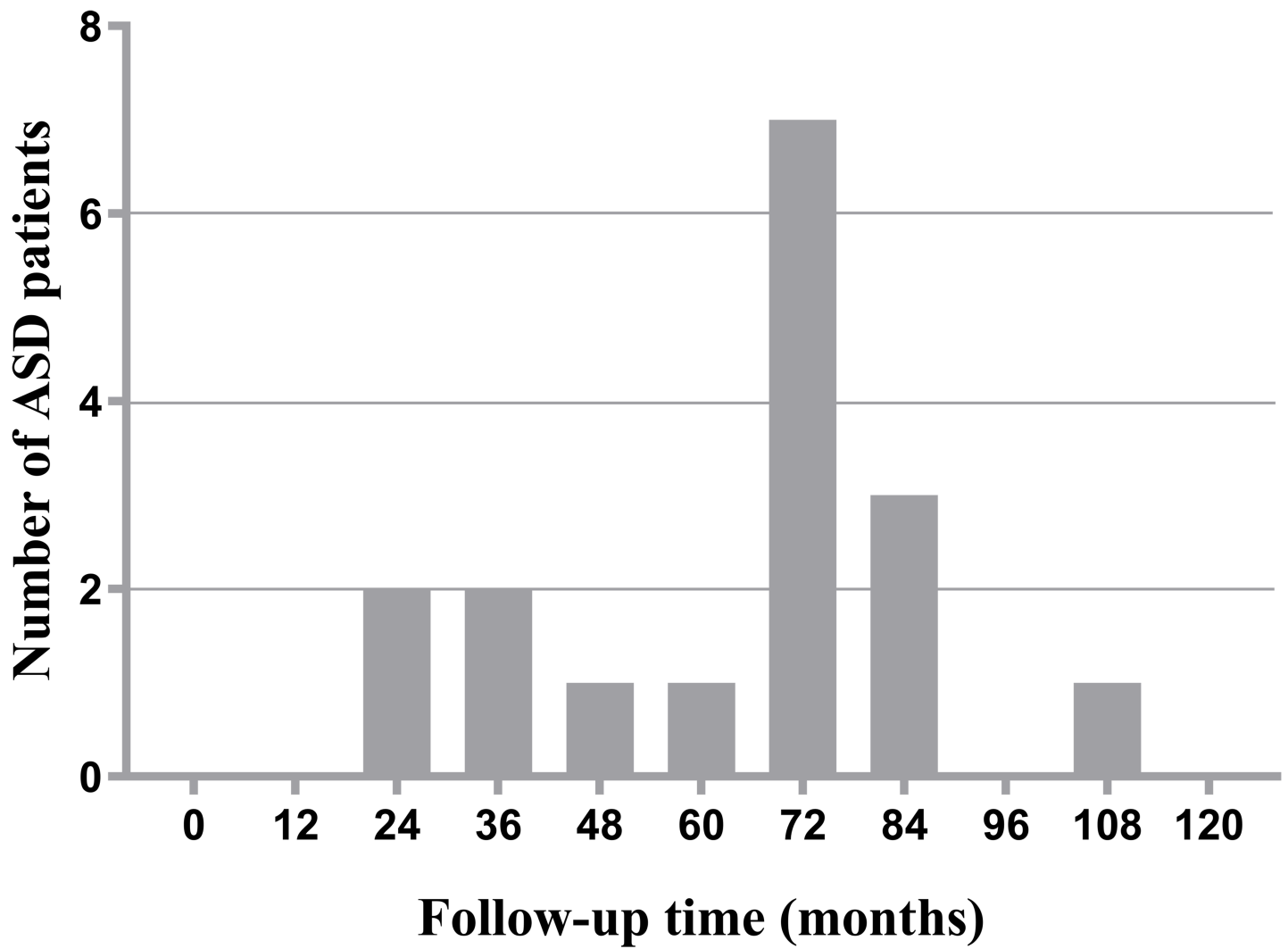
Figure 2

Frequency distribution of follow-up time of adjacent segment disease (ASD）patients after L4-S1 posterior lumbar interbody fusion.

Figure 3

Imaging studies of ASD associated with high postoperative LDI. A, a 47-year-old female patient underwent L4-S1 PLIF with the postoperative LL of 41.79° and LLL of 39.94°.The ultimate LDI equals to 95.57%. B, preoperative sagittal T2-weighted MRI scans at the L3-4 showing no or mild spinal stenosis (Imagama's Classification II) and disc degeneration of Pfirrmann Classification III. C, postoperative neutral lateral X-ray obtained 69 months after surgery revealing retrolisthesis of L3 vertebra. D, the final follow-up MRI scans demonstrating severe spinal stenosis(Imagama's Classification IV) and disc degeneration of Pfirrmann Classification IV
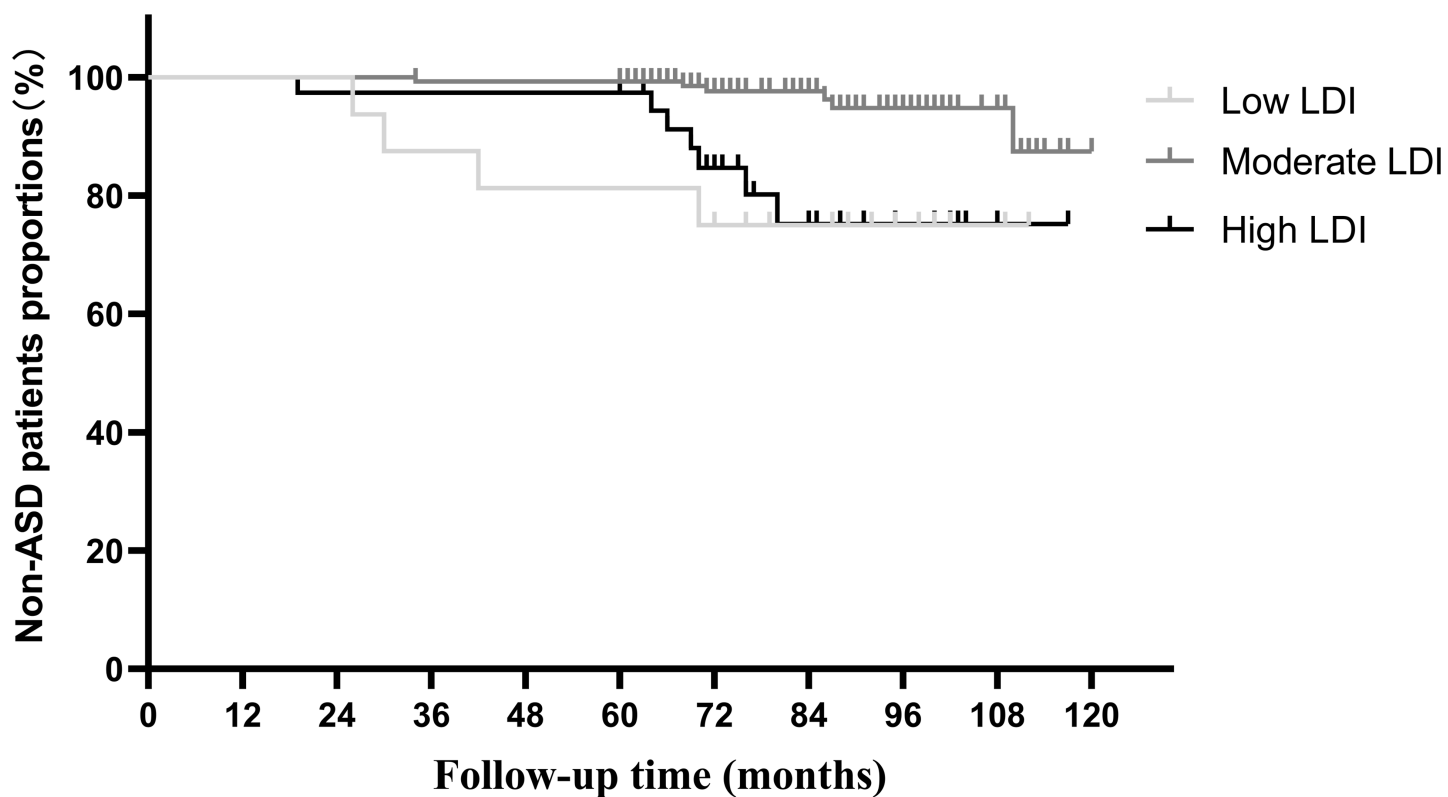
Figure 4

Kaplan-Meier analysis of adjacent segment disease (ASD) patients after L4-S1 posterior lumbar interbody fusion during the 120 months of follow-up period by three kinds of lordosis distribution index (LDI) groups. (Log Rank test, P=0.001)